

conduction in concealed WPW syndrome and the validity as a research and clinical tool to accurately describe atrial activation patterns.

1025-91 P-Wave Configuration in the Signal Averaged Electrocardiogram: Which Filter Technique Differentiates Best Between Patients With Paroxysmal Atrial Fibrillation and Healthy Volunteers?

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Background: Delayed ventricular conduction in the signal averaged ECG is a frequent finding in patients with ventricular tachycardias whereas the atrial conduction delays leading to atrial fibrillation are still a matter of research.

Methods: The aim of this study was to evaluate which of the most commonly used filter techniques (finite impulse response (FIR), least squares fit (LSQ), unidirectional (Uni) and bidirectional (Bidi)) would differentiate best between healthy volunteers and patients with paroxysmal atrial fibrillation (PAF). The signal averaged p-waves during sinus rhythm of 56 patients with PAF were compared to the p-waves of 50 healthy volunteers (N). All recordings were evaluated by two independent observers and the duration of the p-wave was calculated.

Results:

Filter	P duration (N)	P duration (PAF)	p
FIR	109.8 (94-177) ms	133.5 (93-165) ms	0.001
LSQ	114.5 (85-139) ms	126.3 (94-152) ms	0.001
Uni	134 (110-161) ms	142.8 (112-178) ms	0.003
Bidi	102 (73-123) ms	121 (97-141) ms	0.001

Dichotomy limits between N and PAF were: 111 ms (Bidi with sensitivity and specificity of 0.84), 121 ms (FIR: sens. + spec. 0.79), 122 ms (LSQ: sens. + spec. 0.67) and 138.5 ms (Uni: sens. + spec. 0.61).

Conclusion: In comparison to normals, patients with paroxysmal atrial fibrillation showed a significantly prolonged p-wave in the signal averaged ECG. The absolute duration and the dichotomy limits for "healthy" and "PAF" differed largely between applied filter techniques, so did the evaluated sensitivity and specificity values. Bidirectional filtering lead to the clearest differentiation between patients and normals.

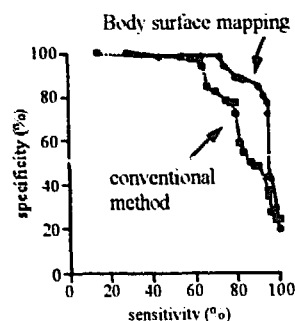
1025-92 Detection of Patients at Risk for Paroxysmal Atrial Fibrillation Improved by Body Surface Mapping of P-Wave Signal-averaged ECG

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P wave signal-averaged ECG (P-SAE) using only orthogonal leads has been useful to detect patients (pts) at risk for paroxysmal atrial fibrillation (Paf). To determine whether the detection accuracy of Paf pts would be enhanced by body surface mapping of P-SAE, we studied 55 Paf pts and 45 control pts. P-SAE was recorded with the P wave-triggering method from the orthogonal leads and 16 precordial unipolar leads. The duration (Ad) of filtered P wave was measured in each of 16 leads to obtain the maximum Ad (Ad[map]). Ad was also measured on the vector magnitude (Ad[vector]). Results are shown below.

	Pat	Control	p value
Ad[map] (ms)	155.5 ± 17.7	128 ± 10.4	p < 0.0001
Ad[vector] (ms)	142.6 ± 16.4	123.5 ± 9.7	p < 0.0001
Ad difference (ms)	12.9 ± 9.4	4.5 ± 5.5	p < 0.0001

Ad difference: the subtraction of Ad[vector] from Ad[map]

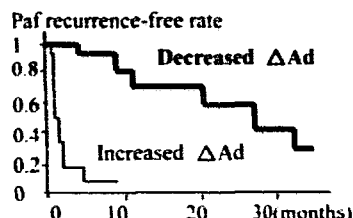


Ad in mapping P-SAE was greater than that in the conventional P-SAE in Paf pts. ROC curve of Ad[map] shifted more upper right sided, in comparison to that of Ad[vector]. When "Ad[map] > 135 ms" and "Ad[vector] > 120 ms" were considered abnormal in each method, the positive predictive value of mapping P-SAE (85%) was significantly (p = 0.01) greater than that of conventional one (85%), while the negative predictive value was comparable (90% vs 85%). Thus, the mapping P-SAE would improve the detectability of Paf pts, in comparison to the conventional method.

1025-93 Prediction of Efficacy of Pilsicainide to Paroxysmal Atrial Fibrillation by Body Surface Mapping of the P-Wave Signal-averaged ECG: A Prospective Study

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We reported that patients (pts) with paroxysmal atrial fibrillation (Paf) would have temporal and spatial dispersion of atrial depolarization increased, by use of body surface mapping of the P wave signal-averaged ECG (P-SAE). To determine whether the efficacy of pilsicainide (PC), a new Class Ic drug, on Paf attacks could be predicted in view of the dispersion, we prospectively studied 25 symptomatic Paf pts (the attack frequency > 1/month). At the entry, P-SAE was recorded by the P wave-triggering method from 16 precordial unipolar leads (V1-V6 in standard ECG and two intercostal spaces upper and lower of V1-V6 except V3) before and one hour after the single dose (100 mg) oral administration of PC. As an index of the dispersion of atrial depolarization, we obtained the difference (ΔAd) between the maximum and minimum of filtered P wave duration in 16 recording sites. These pts were followed up for 10 ± 11 months with PC. In 13 of 25 pts, ΔAd decreased after PC (29.2 ± 10.4 to 20.6 ± 5.5 ms, p < 0.01), while ΔAd increased in the remaining 12 pts (22.9 ± 4.7 to 29.6 ± 9.5 ms, p < 0.01). Paf recurrence documented on ECG was significantly less frequently observed in Paf pts with the decreased ΔAd (46% [6/13]) than those with the increased ΔAd (92% [11/12]) after PC (p < 0.0001).



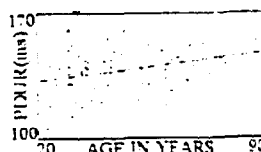
Thus, the efficacy of PC to suppress Paf attacks might be predicted by evaluating the dispersion of filtered P wave duration after the single dose PC administration.

1025-94 Age Dependent Atrial Activation Delay: Observations Using the P-Wave Signal Averaged Electrocardiogram

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The incidence of atrial fibrillation increases with age. The precise explanation is not clear, but age related atrial fibrosis may play a role. We hypothesized that aging would be associated with progressive conduction delay. Atrial activation was assessed by the total P-wave duration (PDur) on signal averaged (SA) ECG in 90 healthy volunteers. Subjects were required to have a normal 12-lead ECG prior to the study and were excluded if hypertension, diabetes, coronary artery disease, congestive heart failure or valvular heart disease were present. P-wave SAECG was performed using the QRS as a trigger and the P-wave as a template to determine PDur from a vector composite of three orthogonal leads.

Results: Using linear regression PDur was significantly correlated with age: $r = 0.32$, $p < 0.002$, $\{PDur = 0.23 (\text{age}) + 126 \text{ ms}\}$ (see figure).



Conclusion: In the absence of known or detectable disease, atrial activa-